Eleven subjects during exposure experienced ear popping, ear or mastoid area pressure, ear pain without infection, or a sensation that the eardrum was moving but not producing a sensation of sound (six males age 2-55 and five females age 19-57). The 2½ year old (A3) pulled on his ears and got cranky repeatedly at the same time as his grandmother's (B2) exacerbations of headache, tinnitus, and ear pain. Five subjects experienced tickling, blowing, or undefined sensations in the external auditory canal, or increased wax production (two men age 42-55 and three women age 52-75).

Balance and equilibrium. Sixteen subjects (seven males age 19-64 and nine females age 12-64) experienced disturbance to their balance or sense of equilibrium during exposure, describing dizziness, light-headedness, unsteadiness, or spinning sensations. One of them, a 42-year-old woman (C2), described how a friend, sitting next to her in her turbine-exposed home, remarked how her eyes appeared to be bouncing back and forth (nystagmus). Ten of these 16 subjects also experienced nausea during exposure to turbines, during or separate from dizziness. No children under the age of 12 had symptoms of dizziness, disequilibrium, or nausea during exposure, except for the usual nausea of acute gastrointestinal and other infections.

Risk factors for dizziness/disequilbrium in the presence of turbines were analyzed using subjects age 12 and up, since this was the youngest age child with this type of symptom. The subject with Parkinson's disease and dementia (E1) was excluded because his baseline balance problems and inability to express himself made it hard for his wife (the informant) to tell if he had worsened symptoms during exposure or not. The remaining sample was 24 subjects. Disequilibrium during exposure is significantly correlated with headaches during exposure ($\chi^2 = 5.08$, $p = 0.024$) and baseline motion sensitivity ($\chi^2 = 4.20$, $p = 0.040$). Disequilibrium during exposure is weakly correlated with tinnitus during exposure ($\chi^2 = 3.60$, $p = 0.054$); inspection of the data shows that these are primarily ataxic (unsteady) subjects. Dizziness/ disequilbrium during exposure is not correlated with VVVD during exposure, pre-existing migraine disorder, previous noise exposure, or prior tinnitus or hearing loss.

Internal quivering, vibration, or pulsation. Eleven adult subjects described these uncomfortable, unfamiliar, and hard-to-explain sensations:

- Dr. J (J1, age 49) described "internal quivering" as part of the "jittery feeling" he has when the turbines are turning fast.

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- Mrs. I (I2, age 52) said the noise inside her house is "low, pulsating, almost a vibration," not shut out by earplugs. She gets a sensation inside her chest like "pins and needles" and chest tightness on awakening at night to noise. "It affects my body – this is the feeling I get when I say I'm agitated or jittery. It's this that gives me pressure or ringing in my ears." "A feeling someone has invaded not only my health and my territory, but my body."

- Mrs. H (H2, age 57) described a pulsation that prevented sleep from the "unnatural" noise from the turbines.

- Mr. G (G1, age 35) described feeling disoriented and "very strange" in certain parts of the house where he could "feel rumbling." If he did not move quickly away from these locations, the feeling would progress to nausea. He described the noise as "at times very invasive. Train noise has a different quality, and is not invasive."

- Mrs. G (G2, age 32) felt disoriented, "light-headed," dizzy, and nauseated in her garden and in specific parts of the house where she detected vibration. She felt her body vibrating "inside," but when she put her hand on walls, windows, or objects, they did not seem to be vibrating.

- Mrs. F (F2, age 51) described a physical sensation of noise "like a heavy rock concert," saying the "hum makes you feel sick."

- Mrs. E (E2, age 56), when supine, felt a "ticking" or "pulsing" in her chest in rhythm with the audible swish of the turbine blades. She interpreted this as her "heart synchronized to the rhythm of the blades," but there is no information (such as a pulse rate from the wrist at the same time) to determine whether this was true or not, or whether she detected a separate type of pulsation. Mrs. E could make these sensations go away by getting up and moving around, but they started again when she lay back down.

- Mr. D (D1, age 64) felt pulsations when he lay down in bed. In addition, "When the turbines get into a particular position (facing me), I get real nervous, almost like tremors going through your body…it's more like a vibration from outside…your whole body feels it, as if something was vibrating me, like sitting in a vibrating chair but my body's not moving." This occurs day or night, but not if the turbines are facing "off to the side."

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- Mr. C (C1, age 45) felt pulsations in his chest that would induce him to hold his breath, fight the sensation in his chest, and not breathe "naturally." Chest pulsations interrupted his sleep and ability to read. He also described a sensation of "energy coming within me…like being cooked alive in a microwave."

- Mrs. B (B2, age 53) described her breath being "short every once in a while, like [while] falling asleep, my breathing wanted to catch up with something."

- Mr. B (B1, age 55) had two episodes of feeling weight on his chest while lying down, which resolved when he stood up. Other than this, he experienced the invasive quality of the noise in his head and ears: "That stuff [turbine noise] doesn't get out of your head, it gets in there and just sits there – it's horrible."

Agitation, anxiety, alarm, irritability, nausea, tachycardia, and sleep disturbance are associated with internal vibration or pulsation:

- Dr. J's (J1, age 49) "jittery" feeling includes being "real anxious," irritable, and "no fun to be around." He interrupts outdoor and family activities to sequester himself in his well-insulated house. When the turbine blades are spinning fast and he detects certain types of noise and vibration as he arrives home from work, he gets queasy and loses his appetite. He awakens from sleep with the "jittery" feeling and tachycardia, and may need to go downstairs to a cot in the 55 degree root cellar (the only place on his property where he cannot hear or feel the turbines) to be able to fall back to sleep. He often takes deep breaths or sighs when in the "jittery" state.

- Mrs. I (I2, age 52) describes episodic "queasiness and nausea" with loss of appetite, "trembling in arms, legs, fingers," "strong mental and physical agitation," and frequent unexpected crying. On noisy nights she awakens after four hours of sleep, weeping in the night. "When I wake up, [there is] more a feeling of pressure and tightness in my chest; it makes me panic and feel afraid." It is "a startling sort of waking up, a feeling there was something and I don't know what it was." Once she awoke thinking there had been an earth tremor (there had not), and twice she has awakened with tachycardia, the "feeling your heart is beating very fast and very loud, so I can feel the blood pumping." Feelings of panic keep her from going back to sleep.

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- Mrs. H (H2, age 57) awakens 5-6 times per night with a feeling of fear and a compulsion to check the house. She describes it as a "very disturbed sort of waking up, you jolt awake, like someone has broken a pane of glass to get into the house. You know what it is but you've got to check it – go open the front door – it's horrific." She finds it hard to fall back to sleep and describes herself as irritable and angry, shouting more at her family members.

- Mr. G (G1, age 35) described the noise outside his home and the noise that awakened him at night "stressful."

- Mrs. G (G2, age 32) was, during exposure, irritable, angry, and worried about the future and her children. She awoke often at night because her children woke up, when she cared for their fears, mentioning none of her own.

- Mrs. F (F2, age 51) described a "feeling of unease all the time." At night she startles awake with heart pounding, a feeling of fear, and a compulsion to check the house. The feeling of alarm keeps her from being able to go back to sleep.

- Mrs. E (E2, age 56) did not express anxiety or fear, but she awakened repeatedly at night and was unable to get back to sleep on nights when the turbines were facing the house.

- Mr. D (D1, age 64) described how he has to "calm down" from the "tremor." If outside, "I come in, sit down in my chair and try to calm myself down. After an episode like that, I'm real tired." Mood has worsened with increased anger, frustration, and aggression. Tachycardia accompanies the "tremor" at times: "My heart feels like it's starting to race like crazy and I have these tremors going through my body." Mr. D pants or hyperventilates when the tremor and tachycardia occur, and consciously slows his breathing when calming down.

- Mr. C (C1, age 45) was unable to rest, relax, or recuperate in his home, where his body was "always in a state of defense." He had to drive away in his car to rest.

- Mrs. B (B2, age 53) became "upset and in a turmoil" when her symptoms were worse, leaving her house and tasks repeatedly to get relief.

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- Mr. B (B1, age 55) described stress, "lots, pretty near more'n I could take, it just burnt me, the noise and run-around." He was prescribed an anxiolytic, and spent more time at the shore and his boat for symptom relief.

The internal quivering, vibration, or pulsation and the associated complex of agitation, anxiety, alarm, irritability, tachycardia, nausea, and sleep disturbance together make up what I refer to as *visceral vibratory vestibular disturbance* (VVVD). Fourteen adult subjects (six men age 35-64 and eight women age 32-75) had VVVD during exposure, including the eleven quoted above and Mr. F (F1, age 42), Mrs. F Senior (F4, age 75), and Mrs. C (C2, age 42). Mr. I (I1, age 59) had partial symptoms, with urge to escape, noise-induced nausea, and sleep disturbance, but no feeling of internal movement. VVVD resolves immediately upon leaving the vicinity of the turbines, when the turbines are still and silent, and under favorable weather conditions at each locality.

Because VVVD is in part a panic attack, accompanied by other physical and mental symptoms, I examined the relationships among VVVD and panic disorder, other mental health diagnoses, and other risk factors. The sample for this analysis was 21 adults ages 22 and above (since the study had no participants age 22-29, this is the same for this study as starting with the age group of the youngest symptomatic subjects, who were 32).

No study subjects had pre-existing panic disorder or previous isolated episodes of panic, so there was no correlation between pre-existing panic and VVVD. Seven subjects had immediate pre-exposure (2 subjects) or past histories (5 subjects) of mental health disorders including depression, anxiety, post-traumatic stress disorder (PTSD), and bipolar disorder. There was no correlation between immediate pre-exposure or past mental health disorder and VVVD ($\chi^2 = 0.429$, $p = 0.513$). There was, however, a highly significant correlation between VVVD and motion sensitivity ($\chi^2 = 7.88$, $p = 0.005$).

There was also a moderately significant correlation between VVVD and headaches during exposure ($\chi^2 = 4.95$, $p = 0.026$). There was no correlation between VVVD and dizziness or tinnitus during exposure, or between VVVD and pre-existing migraine, tinnitus, or hearing loss.

Concentration and memory. Twenty of the 34 study subjects age 4 and up (eleven males age 6-64 and nine females aged 5-56) had salient problems with concentration or memory during exposure to wind turbines compared to pre- and/or post-exposure. This is a conservative count, including only subjects whose accounts included specific information on decline in school and homework performance (for

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children and teens) or details on loss of function for adults. Eight other subjects had some disturbance to concentration and memory, but symptoms were milder or the descriptions more vague (in their own or parents' accounts). Five others subjects, all older adults, noted no change compared to pre-existing memory problems. This leaves only one subject, a 19-year-old woman home from college and minimally exposed (B3), who did not have baseline deficits and was unaffected.

Pre-exposure cognitive, educational, and work accomplishments, specific difficulties related to concentration and memory during exposure, and degree and timing of post-exposure recovery are documented in the Family Tables for each individual, under "Cognition." Difficulties are often striking compared to the subject's usual state of functioning:

- Mr. A (A1, age 32), a professional fishermen with his own boat, who had an isolated difficulty with memory for names and faces prior to exposure, became routinely unable to remember what he meant to get when he arrived at a store, unless he had written it down.

- Mrs. B (B2, age 53), a homemaker, got confused when she went to town for errands unless she had written down what she was going to do, and had to return home to get her list. When interviewed six weeks after moving, she reported that she had improved to being able to manage three things to do without a list.

- Mr. C (C1, age 45) had to put reading aside because he could not concentrate whenever he felt pulsations.

- Mrs. C (C2, age 42), a very organized mother of six who was "ready a month in advance for birthday parties" prior to exposure, became disorganized and had difficulty tracking multiple tasks at once, including while cooking, repeatedly boiling the water away from pots on the stove. She remarked, "I thought I was half losing my mind."

- Mr. D (D1, age 64), a disabled, retired industrial engineer, noticed progressive slowing of memory recall speed and more difficulty remembering what he had read.

- Mrs. E (E2, age 56), a retired teacher active in community affairs, could not spell, write e-mails, or keep her train of thought on the telephone when the turbine blades were turned towards the house, but was able to do these things when the blades were not facing the house.

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- Mrs. F (F2, age 51), a nurse, child development specialist, midwife, and Masters level health administrator, could not follow recipes, the plots of TV shows, or furniture assembly instructions during exposure.

- Mrs. G (G2, age 32), a well-organized mother of four, was forgetful, had to write everything down, could not concentrate, and could not get organized. She forgot a child's hearing test appointment. She did not have memory or concentration problems during a previous depression at age 18, and described her experience as "different this time."

- Mr. I (I1, age 59), a professional gardener, could not concentrate on his outdoor gardening and building tasks if the turbines were noisy, saying "after half an hour you have to leave, escape, close the door."

- Dr. J. (J1, page 49), a physician, noticed marked concentration problems when he sat down to pay bills in a small home office with a window towards the turbines.

Decline in school performance compared to pre-exposure, or marked improvement in school performance after moving away from turbines, was noted for 7 of the 10 study children and teens attending school (age 5-17; C7, F3, G3, G4, H3, J3, J4). For example:

- A 17-year-old girl (F3), a diligent student, was not concerned about the turbines and thought her parents were overdoing their concern until she unexpectedly did worse on national exams than the previous year, surprising her school, family, and self. At this point she began accompanying her parents to their sleeping house.

- A 9-year-old boy (C7), whose school work was satisfactory without need for extra help prior to exposure, failed tests, lost his math skills, and forgot his math facts. He could not maintain his train of thought during homework, losing track of where he was if he looked up from a problem.

- A 6-year-old boy (G3), described as an extremely focused child and advanced in reading prior to exposure, did not like to read during exposure. Two months post-exposure, now age 7, he would sit down to read on his own for an hour at a time, reading "quite a thick book" for his age.

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- His 5-year-old sister (G4) had a short attention span prior to exposure. Her hearing loss due to bilateral chronic serous otitis media was thought to be interfering with school work during exposure, and she repeatedly had tantrums over schoolwork at home during the exposure period. Two months after moving, despite no change in her ears (on a waiting list for pressure equalization tubes), she was more patient and could work longer on homework. Her mother noted that her "schoolwork has improved massively."

- An 8-year old boy (H3) had an excellent memory and did well in reading, spelling, and math prior to exposure. During exposure he became resistant to doing homework, with tantrums, and his teacher told him he was not concentrating and needed to go to bed earlier.

In comparing the 20 subjects with salient concentration or memory changes to the 14 who had no change from baseline or vague/minimal difficulties, there are significant relationships with 1) baseline cognition, in that those without memory or concentration deficits at baseline are more likely to notice such deficits during exposure ($\chi^2 = 4.86$, $p = 0.027$); and 2) fatigue or loss of energy or enjoyment for usual activities during exposure ($\chi^2 = 5.61$, $p = 0.018$). There is no significant relationship between salient concentration or memory changes and pre-existing psychiatric diagnoses, migraine, motion sensitivity, or noise exposure, or between salient concentration or memory changes and headache, tinnitus, VVVD, or irritability during exposure.

In addition to the statistical association between fatigue and concentration disturbance, a number of subjects directly attributed their concentration problems to their sleep deprivation or disturbance. Several aspects of the data, however, suggest that other factors may also be involved.

First, one subject, Mrs. E (E2, age 56) could not do certain mental tasks requiring concentration when the turbines were turned towards her house, but could do them when the turbines were not turned towards the house. Mr. C (C1, age 45), Mr. I (I1, age 59), and Dr. J (J1, age 49) also had concentration problems closely linked in time and space to direct exposure to turbine noise.

Second, some of the problems described by subjects, such as Mrs. F (F2, age 51) and the members of families A and B, are more extreme than I expect from sleep deprivation. The degree of thinking dysfunction involved in not being able to follow a recipe or assemble a piece of furniture, in a woman both highly educated and involved in several practical professions (nursing and farming), does not match

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my expectation of sleep deprivation from the experience, for example, of both younger and older physicians, who often function under sleep deprivation.

Third, some subjects had concentration problems without obvious sleep problems. All four members of family J had concentration problems, but only Dr. J (J1, age 49) was sleep deprived. Mrs. J (J2, age 47) fell asleep easily and usually went back to sleep if awakened, but still had problems with memory and focus in her home activities that she had noticed and attempted to treat. Their 13-year-old son (J3) needed white noise or music to drown out turbine noise to fall asleep, but went to sleep promptly, slept through the night, and did not complain in the morning of being tired or having slept poorly. His school performance and his level of distractibility at home, however, were both markedly different than at baseline. The younger son, age 8 (J4), continued to sleep well, but still had a surprising decline in school performance, though milder and of shorter duration than his brother's.

Fourth, the problems with concentration and memory resolve on a different schedule from the turbine-related sleep problems. Sleep problems resolve immediately except when accompanied by persistent depression (C1, F1). Problems with concentration and memory frequently took longer to improve, even in the absence of depression. To study resolution, we need to look at subjects who have moved away from their exposed homes or spent a prolonged period away that included work (families A, B, C, E, F, and G, and Mrs. I), since vacations do not provide the same challenges to concentration and memory. Of these 23 subjects over age 4, 13 had salient difficulties with concentration or memory:

- Mr. A (A1, age 32) rated his memory as 85% at baseline, 2% during exposure, and 10% six weeks after moving away.

- Mr. and Mrs. B (B1, B2, age 55 and 53) said their memories had partially recovered six weeks after moving.

- Mr. C (C1, now age 47), with continuing depression and ongoing exposure for house maintenance, noted 25 months after moving how bad his memory seemed.

- Mrs. C (C2, now age 44) felt she had recovered her memory and concentration 18 months after moving, despite ongoing stress from crowded living arrangements. Her affected son (now age 11, C7) had not completely recovered his school performance.

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- Mrs. E (age 52) recovered immediately. She only experienced problems during exposure when the turbines were turned in a particular direction.

- Mr. and Mrs. F (F1, F2, ages 42 and 51) had moved away but still worked at their turbine-exposed home and farm during the day. Three months after they moved, both thought their concentration had improved, but not to baseline. Mr. F, with ongoing depression, did not perceive any memory recovery. I do not have information about their daughter's (F3, age 17) exam performance after moving.

- Mrs. G (G2, age 32) rated her memory as 10/10 at baseline, 2/10 during exposure, and 5/10 two months after moving away, at which point her depression was mostly resolved. Mrs. G's 5-year-old and 6-year-old children (G3, G4) showed marked improvements in concentration by two months after moving.

Only three subjects were clearly depressed during or after exposure. Mrs. G (G2, age 32) was becoming depressed at the time of the first (during exposure) interview. She remarked on the difference in her cognitive functioning between her current experience and a previous episode of depression at age 18, when she had no problem with her memory or concentration. Two other subjects, Mr. C (C1, age 45) and Mr. F (F1, age 42) developed depression after they had to abandon their homes, which was associated with prolonged memory difficulties. Both also had ongoing exposure.

Irritability and anger. Twenty-eight subjects (fifteen male age 2-64 and thirteen female age 2-64) perceived themselves or were noted by parents to be more angry, irritable, easily frustrated, impatient, rude, defiant, or prone to outbursts or tantrums than at baseline. The adults were uniformly apologetic about their own irritability, and several described how careful they were to avoid acting irritable in their households. Four children (three boys age 8-9 and a girl age 5; C7, G3, H3, G4) were markedly frustrated over homework. The young children of family G quarreled and had tantrums incessantly, and the six children/young adults in family C became angry, prickly, moody, defiant, or prone to fights at school. In families with children, the breakdown in children's behavior, social coping skills, and school performance was one of the strongest elements propelling them to move.

Fatigue and motivation. Twenty-one subjects felt or acted tired, and 24 had problems with motivation for usual, necessary, or formerly enjoyable activities (27 combined, fourteen male age 2-64 and thirteen female age 2-75). Like concentration and memory, these symptoms undoubtedly have a relationship with

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sleep deprivation, but certain subjects described leaden feelings around turbines that resolved as soon as they left the vicinity, such as Mr. A (A1, age 32), who said, "You feel different up there: draggy, worn out before you even start anything.... It was a chore to walk across the yard." After driving an hour away to visit a family member, "I felt better all over, like you could do a cart wheel," and he felt well after moving.

When away from their turbine-exposed homes, most subjects recovered their baseline positive mood states, energy, and motivation immediately. Six adult subjects did not. These were Mr. B (B1, age 55), Mr. and Mrs. C (C1, C2, age 45 and 42), Mr. and Mrs. F (F1, F2, age 42 and 51), and Mrs. G (G2, age 32). By their own accounts, three (Mr. C, Mr. F, and Mrs. G) had unresolved or resolving depression. All but Mrs. G had ongoing anxiety and anger over abandoning their homes and their unresolved life situations.

Other symptom clusters and isolated problems

These symptoms and problems occurred in fewer subjects and typically require more than a medical history to diagnose. Several are exacerbations of pre-existing conditions with obvious connections to situations of high stress (cardiac arrhythmias, hypertension, irritable bowel, gastroesophageal reflux, glucose instability). Others are sequelae of core symptoms (auditory processing problems, unusual migraine aura). Others may indicate different kinds of direct effects of noise on body tissues, as in the vibroacoustic disease model of noise effects (respiratory infections, asthma, clotting abnormalities),[32] or other types of secondary effects (asthma).[33]

Respiratory infection/inflammation cluster: Seven subjects had unusual or prolonged lower respiratory infections during exposure (A2, B1, C2, E2, F1, F3, F4), and two of these also had prolonged asthma exacerbations (F1, F3). Four subjects had unusually severe or prolonged middle ear problems (C7, F2, G3, G4).

Cardiovascular cluster: Two subjects had exacerbations of preexisting dysrhythmias (F1, J2). Two women had hypertension that increased during and after the exposure period, requiring medication after

[32] Castelo Branco NAA , Alves-Pereira M. 2004. Vibroacoustic disease. Noise Health 6(23): 3-20.
[33] Beasley R, Clayton T, Crane J, von Mutius E, Lai CK, Montefort S, Stewart A; ISAAC Phase Three Study Group. 2008. Association between paracetamol use in infancy and childhood, and the risk of asthma, rhinoconjunctivitis, and eczema in children aged 6-7 years: analysis from Phase Three of the ISAAC programme. Lancet 372(9643): 1039-48.

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the end of the exposure period. Both still had considerable stress related to moving out and not being able to establish another regular home, and depressed husbands (C2, F2).

Gastrointestinal cluster. Four subjects had exacerbations of pre-existing gastroesophageal reflux (GER), ulcer, or irritable bowel, two with irritable bowel and upper gastrointestinal symptoms at the same time (D1, F1, F2, J2).

Arthralgia/ myalgia cluster. One healthy 32-year-old woman (G2) noted pain in one elbow while in her exposed house. It resolved when she went away for vacations with her family, and recurred when she returned. It resolved quickly when the family moved away, even though she did lots of lifting during the move. Two women (age 56-57; E2, H2) had exacerbations of fibromyalgia, both of which resolved after moving or during times away from their exposed home.

Diabetes control. A 56-year-old man with Type II diabetes (E1), stable on oral medications and insulin before exposure, had marked glucose instability accompanied by visual blurring, retinal changes, and polyuria during exposure.

Anticoagulation: A 75-year-old woman with atrial fibrillation (F4) had stable INR values on 2-4 mcg warfarin daily for 10 years. By 16 months of exposure, her warfarin dose had been increased to 8-9 mcg daily in response to decreasing INR values.

Auditory processing cluster: A woman (age 33, A2) had progressively worsening tinnitus during her five months of exposure. After she moved away, the tinnitus resolved and she noticed she had a new difficulty understanding conversation in a noisy room, now needing to watch the speaker's face carefully. Her son, age 27-32 months during exposure, did not confuse sounds before exposure but began to do so during exposure, and continued to do so when I interviewed mother six weeks after the exposure ended (A3). The child's language development was otherwise good. One woman (age 42, C2) had tinnitus throughout her 21 month exposure period without hearing changes. After she moved and the tinnitus resolved, she noted hyperacusis. Another woman (age 32, G2) experienced hyperacusis during exposure, but no tinnitus. The hyperacusis resolved after the family moved.

Ocular cluster: Three subjects exposed to the same turbines (two men age 32-55 and one woman age 53; A1, B1, B2) had ocular pain, pressure, and/or burning synchronously with headache and tinnitus. Mr. D

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(D1, age 64) had a painless retinal stroke, losing half the vision in his left eye. Mr. D had a normal CT scan of the brain and was examined by an ophthalmologist.

Complex migraine phenomena. A 19-year-old fisherman (C4) with migraine at baseline had complex visual symptoms with flashes in square patterns in one eye at a time (scintillating scotoma), evolving to blurring and visual loss for 30 seconds to 2 minutes, also in one eye at a time (amaurosis fugax), right more than left, repetitively during the last month of his 15-21 month exposure until 8-12 months after exposure ended, with a decrease in frequency by 7 months after moving out. These events happened at any time of day and rarely overlapped with headaches or tinnitus. He had normal ophthalmologic exams, normal MRI and MRA scans of the brain and associated arteries, and a normal evaluation for clotting abnormalities and vasculitis. The events resolved completely with normal vision. The same man experienced repetitive complex basilar migraines with aura after the first few months of his 15-21 month turbine exposure, involving daily bilateral paresis and paresthesias of his legs and occasional headache, tinnitus, and light-headedness. The leg symptoms resolved on the same schedule as the eye symptoms, though headaches and nausea continue to be triggered regularly by seasickness.

Discussion

The core symptoms of Wind Turbine Syndrome are sleep disturbance, headache, tinnitus, other ear and hearing sensations, disturbances to balance and equilibrium, nausea, anxiety, irritability, energy loss, motivation loss, disturbances to memory and concentration, and *visceral vibratory vestibular disturbance* (VVVD). Core symptoms are defined as common and widely described by study participants, closely linked in time and space to turbine exposure, and amenable to diagnosis by medical history. The latter was a particular requirement of this study. The subjects of this study had other types of health problems during exposure, discussed in "Other symptom clusters and isolated problems," but different types of study will be needed to find out if there is a link between these problems and wind turbine exposure.

The most distinctive feature of Wind Turbine Syndrome is the group of symptoms I call *visceral vibratory vestibular disturbance*, or VVVD. The adults who experience this describe a feeling of internal pulsation, quivering, or jitteriness, accompanied by nervousness, anxiety, fear, a compulsion to flee or check the environment for safety, nausea, chest tightness, and tachycardia. The symptoms arise day or night, interrupting daytime activities and concentration, and interrupting sleep. Wakefulness is prolonged after this type of awakening. Subjects observe that their symptoms occur in association with specific

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types of turbine function: the turbines turned directly towards or away from them, running particularly fast, or making certain types of noise. The symptoms create aversive reactions to bedroom and house. Subjects tend to be irritable and frustrated, especially over the loss of their ability to rest and be revitalized at home. Subjects with VVVD are also prone to queasiness and loss of appetite even when the full set of symptoms is not present.

There is no statistical association in this study between VVVD and pre-existing panic episodes (which occurred in none of the subjects) or other mental health disorders, such as depression, anxiety, bipolar disorder, or posttraumatic stress disorder. There is a highly significant association between VVVD and pre-existing motion sensitivity ($p = 0.005$).

Headaches more frequent or severe than at baseline occurred in all migraineurs in the study, and all children with headaches in the study were migraineurs or the children of migraineurs. Non-migrainous adults also got severe headaches around turbines, and indeed about half the people with headache worse than baseline (9 out of 19) were adults without history of migraine. Pre-exposure migraine is a significant risk factor for more severe or frequent headaches during turbine exposure ($p = 0.004$), but does not account for all the cases of headache.

Tinnitus occurred as a migraine aura in three subjects, but statistically in the study group tinnitus was not significantly associated with migraine, but rather with previous industrial noise exposure ($p = 0.013$), past history of tinnitus ($p = 0.017$), and baseline permanent hearing impairment ($p = 0.040$). I interpret tinnitus in these non-migrainous subjects as the direct impact of turbine noise on the cochlea, sensitized by previous inner ear damage from earlier noise exposure or chemotherapy.

Visceral vibratory vestibular disturbance (VVVD)

The work of Mittelstaedt on visceral detectors of gravity,[34] and Balaban and others on balance-anxiety linkages,[35,36,37,38,39] opens a window on the VVVD symptom set. Balaban, a neuroscientist, has localized

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[36] Balaban CD. 2002. Neural substrates linking balance control and anxiety. Physiology and Behavior 77: 469-75.
[37] Furman JM, Balaban CD, Jacob RG. 2001. Interface between vestibular dysfunction and anxiety: more than just psychogenicity. Otol Neurotol 22(3): 426-7.
[38] Balaban CD. 2004. Projections from the parabrachial nucleus to the vestibular nuclei: potential substrates for autonomic and limbic influences on vestibular responses. Brain Res 996: 126-37.
[39] Halberstadt A, Balaban CD. 2003. Organization of projections from the raphe nuclei to the vestibular nuclei in rats. Neuroscience 120(2): 573-94.

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and described the neural connections among the vestibular organs of the middle ear, brain nuclei involved with balance processing, autonomic and somatic sensory inflow and outflow, the fear and anxiety associated with vertigo or a sudden feeling of postural instability, and aversive learning.[40] These form a coordinated, neurologically integrated system based in the parabrachial nucleus of the brainstem and an associated neural network.[41,42] Several aspects of this system need to be considered here.

First, there appear to be not three but four body systems for regulating balance, upright posture, and the sense of position and motion in space. The first three systems are the eyes, the semicircular canals and otolith organs of the inner ear, and somatic input from skin, skeletal muscles, tendons, and joints. The fourth system is visceral detection of gravity and acceleration (meaning change in speed or direction of movement) by visceral graviceptors. These include stretch receptors in mesenteries or other connective tissue supporting organs or great vessels, and integrated systems of pressure detection in vessels and organs.[43] Such receptors have been found in the kidneys and in structures supporting the great vessels in the mediastinum, among other locations.[44] Von Gierke (an older dean of vibration studies for the US space program) considers the inter-modality sensory conflict between the abdominal visceral graviceptors and the otolith organs to be a possible cause of motion sickness.[45]

The second critical element is central processing: how sensory information about motion and position is integrated by the brain, what other brain centers are activated, and what kinds of signals the brain then sends back out to the body. Balaban and colleagues describe how the parabrachial nucleus network receives motion and position information from visual, vestibular (inner ear), somatosensory, and visceral sensory input, and is linked to brain centers and circuits that mediate anxiety and fear, including the amygdala, a key mediator of fear reactions, and serotonin and norepinephrine-bearing neurons radiating from the midbrain.[46,47,48] Our sense of balance and stability in space is closely connected – neurologically – to fear and anxiety.

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[40] Balaban and Yates 2004
[41] Balaban CD, Thayer JF. 2001. Neurological bases for balance-anxiety links. J Anx Disord 15: 53-79.
[42] Balaban 2002
[43] Balaban and Yates 2004
[44] Vaitl D, Mittelstaedt H, Baisch F. 2002. Shifts in blood volume alter the perception of posture: further evidence for somatic graviception. Int J Psychophysiol 44(1): 1-11.
[45] von Gierke HE, Parker DE. 1994. Differences in otolith and abdominal viscera graviceptor dynamics: implications for motion sickness and perceived body position. Aviat Space Environ Med 65(8): 747-51.
[46] Balaban and Thayer 2001
[47] Balaban 2002
[48] Halberstadt and Balaban 2003.

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Balaban illustrates with a story. He asks the reader to visualize waiting in traffic on a hill for a light to turn. Out of the corner of your eye you see the truck next to you starting to inch forward, and you jam your foot on the brake, since your sensory system has told you that you are starting to slip backwards. There's a bit of panic in that moment, quickly settled as you realize you are indeed stable in space and not moving. The story illustrates how a sensation of unexpected movement elicits alerting and fear. When the sense of movement is ongoing and cannot be integrated with the evidence of the other senses, as happens in vertigo, there is a more prolonged fear reaction. The association of fear with vertigo has been known since ancient times.[49]

The third critical element is integrated neurologic outflow to the body from the parabrachial nucleus network to both the somatic (conscious, voluntary) and visceral (autonomic) effector systems. The somatic musculature is responsible for that fast foot on the brake, for righting movements of limbs, torso, and neck; and for breathing motions of the diaphragm and chest wall. The autonomic system is responsible for blood flow, heart rate, blood pressure, sweating, nausea, and other automatic, non-conscious modifications to visceral functioning. In a fear response, there is integrated outflow to these two systems. The parabrachial nucleus network is also involved in aversive learning, [50] an experience in which nausea, if present, plays a large role.

In VVVD, subjects detect unusual types of movement (pulsation, internal vibration, internal quivering) or other sensations (pressure, a sense of fighting something to breathe, pins and needles) in the chest or in the coordinated chest-abdominal internal space. The chest and abdomen are separated and unified by the diaphragm, which, as a striated somatic muscle, has fine-grained sensitivity to motion and stretch. The diaphragm sends signals to the brain which are specific and localizable in time and space, as opposed to visceral receptors, which send signals that are vague, like discomfort, malaise, fullness, or nausea. The diaphragm is tightly bound to one of the largest abdominal organs, the liver, and they move as a unit during breathing.

The chest, via the mouth, nose, trachea, smaller airways, and air sacs of the lungs, is open to the air. Pressure fluctuations in the air (sound waves) have free access to this airspace within the body when we breathe. Pressure fluctuations in the air also have access to the ear, which is designed to funnel them to the tympanic membrane, which concentrates their energy and transmits it to the inner ear. The ear and the

[49] Balaban and Thayer 2001
[50] Balaban and Thayer 2001

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chest are different size spaces with walls of different mobility and elasticity. Hence they respond differently to air pressure fluctuations (sound waves) of different sizes.

Studies of whole body vibration focus on the easily mobile diaphragm and coupled abdominal organs. Being mobile, with the air of the lungs on one side and the soft abdominal wall on the other, this thoraco-abdominal system is easily set in motion by lower energy (amplitude) vibrations than are required to perturb other parts of the body.[51] Each part of the body has its own resonance frequency with regard to vibration. When an object is vibrated at its resonance frequency, the vibration is amplified. The resonant frequency of the thoraco-abdominal system, as it moves vertically towards and away from the lungs, lies between 4 and 8 Hz for adult humans.[52] Vibrations between 4 and 6 Hz set up resonances in the trunk with amplification up to 200%.[53] Related chest and abdominal effects are found in the same frequency range. Vibrations in the 4-8 Hz range influence breathing movements, 5-7 Hz can cause chest pains, 4-10 Hz abdominal pains, and 4-9 Hz a general feeling of discomfort.[54] In small children under 40 pounds, the vertical resonance or power absorption peaks at 7.5 Hz, as opposed to 4-5 Hz for adults.[55]

Low frequency noise can cause the human body to vibrate, as quantified by researchers in Japan.[56] The degree to which the body surface is induced to vibrate by low frequency noise is correlated with subjective unpleasantness (a sensation suggesting visceral as well as surface/somatic stimulation by the noise) .[57]

With this background, I hypothesize the following mechanism for VVVD. Air pressure fluctuations in the range of 4-8 Hz, which may be harmonics of the turbine blade passing frequency, may resonate (amplify) in the chest and be felt as vibrations or quivering of the diaphragm with its attached abdominal organ mass (liver). Slower air pressure fluctuations, which could be the blade passing frequencies themselves or a low harmonic (1-2 Hz), would be felt as pulsations, as opposed to the faster vibrations or quivering. (The vibrations or pressure fluctuations may also be occurring at different frequencies, without resonance amplification.) The pressure fluctuations in the chest could disturb visceral receptors, such as large vessel or pulmonary baroreceptors or mediastinal stretch receptors, which function as visceral

[51] Coermann RR, Ziegenruecker GH, Wittwer AL, von Gierke HE. 1960. The passive dynamic mechanical properties of the human thorax-abdominal system and of the whole body system. Aerospace Medicine 31(6): 443-55.
[52] von Gierke and Parker 1994
[53] Hedge, Alan, 2007
[54] Rasmussen 1982
[55] Giacomin J. 2005. Absorbed power of small children. Clin Biomech 20(4): 372-80.
[56] Takahashi Y, Yonekawa Y, Kanada K, Maeda S. 1999. A pilot study on the human body vibration induced by low-frequency noise. Industrial Health 37: 28-35.
[57] Takahashi Y, Kanada K, Yonekawa Y, Harada N. 2005. A study on the relationship between subjective unpleasantness and body surface vibrations induced by high-level low-frequency pure tones. Industrial Health 43: 580-87, p. 580.

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graviceptors. These aberrant signals from the visceral graviceptors, not concordant with signals from the other parts of the motion-detecting system, have the potential to activate the integrated neural networks which link motion detection with somatic and autonomic outflow, emotional fear responses, and aversive learning. The people who are susceptible to responding in this way are those who in the past have become nauseated in response to other vertically oriented, anomalous environmental movements (seasickness or carsickness). Thus panic episodes with autonomic symptoms such as tachycardia and nausea arise during wakefulness or sleep in people with pre-existing motion sensitivity but without prior history of panic, anxiety, or other mental health disorders. Repeated triggering of these symptoms creates aversive learning, wherein the person begins to feel horror and dread of things associated with the physical sensations, such as his bedroom or house where he previously found comfort and regeneration.

VVVD was identified in the study in 14 out of 21 adult subjects. The behavior and experiences of other subjects, especially children, could be interpreted as partial manifestations of the same problem. For example, the two toddlers in the study, both aged 2½ (A3, G5), had night terrors. They awoke screaming multiple times per night, and were inconsolable and difficult to get back to sleep. The little girl (G5) would fight her mother, grabbing onto the posts of the bunk bed, to avoid going back into her own bed after awakening in this state. This shows clear parallels with the fear responses, prolonged awake periods, and aversive responses of the adults with VVVD. Both toddlers were agitated and irritable in the daytime, also similar to the adults in the study. Both 5-year-olds in the study, a boy and a girl (C7, G4), also frequently woke up fearful at night.

Perturbing the inner ear

I propose that disrupted stimulation of other channels of the balance system, especially the inner ear vestibular organs, is also likely to play a role in Wind Turbine Syndrome. Altogether, in subjects with or without VVVD, the Wind Turbine Syndrome core symptoms resemble the symptoms of a balance or vestibular disorder, meaning malfunctioning of the inner ear motion-detecting organs (peripheral vestibular dysfunction) or of brain processing of balance-related neural signals (central balance dysfunction). Near wind turbines, I suggest that these symptoms may arise through disturbed input to the classical pathways of motion and position perception (the visual, vestibular, and somatosensory channels), perhaps in an additive fashion. If several pathways are abnormally stimulated at the same time, it becomes even more likely that multisensory vestibular brain centers[58] will receive disordered or

[58] Dieterich M, Brandt T. 2008. Functional brain imaging of peripheral and central vestibular disorders. Brain, E-pub ahead of print, May 30, 2008, pp. 1-15.

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including humans. These structures are used by fish (like us) to sense acceleration and tilt relative to gravity, but also to detect nearby perturbations in the water ("near-field sound") with peak sensitivities in the low frequency range between 40 and 120 Hz.[66] Certain fish are also known detect distant low-frequency sound, which they use to navigate relative to distant shores where waves are breaking.[67]

Most exciting, new research provides direct experimental evidence that normal human vestibular organs are sensitive to low-frequency vibration (which is the same as bone-conducted sound) and *much more sensitive than the cochlea.*[68] Among normal subjects, this sensitivity is "tuned" (has a sharp peak of sensitivity and response) at 100 Hz (G-G# 1½ octaves below middle C, keys 23-24 on a piano). Researchers applied carefully calibrated force and vibration frequency directly to the bony mastoid prominence behind the subjects' ears. They were able to elicit and measure responses of the vestibulo-ocular reflex at vibration intensities 15 dB below the subjects' hearing thresholds. In other words, the subjects could no longer hear the bone-conducted sound created by applying vibration to their mastoids, but the vestibular parts of the inner ear still picked up the vibration and transmitted signals into the balance and motion networks in the brain, resulting in specific types of eye muscle activation. Since dB is a base 10 logarithmic measure, *15 dB below* means a signal 0.0316 ($10^{-1.5}$), or about 3%, of the power or amplitude of the signal these normal subjects could hear.

The researchers note that "the very low thresholds we found are remarkable as they suggest that humans possess a frog- or fish-like sensory mechanism which appears to exceed the cochlea for detection of substrate-borne low-frequency vibration and which until now has not been properly recognized."[69] Thus the potential exists, in normal humans, for stimulation and disruption of balance signals from the inner ear by low-frequency noise and vibration, even when the noise or vibration does not seem especially loud.

Central balance processing

When there is conflict in healthy people among the signals coming from the different balance channels, the brain areas that integrate the information quickly compensate by suppressing or down-weighting the anomalous channel.[70] On functional brain scans, vestibular and visual cortical areas show a pattern of

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[66] Fay RR, Simmons AM. 1999. The sense of hearing and fishes and amphibians. In *Comparative Hearing: Fish and Amphibians*, ed. Fay RR, Popper AN, pp. 269-317. Springer-Verlag, New York.
[67] Sand O, Karlsen HE. 1986. Detection of infrasound by the Atlantic cod. J Exp Biol 125: 197-204.
[68] Todd NPMc, Rosengren SM, Colebatch JG. 2008. Tuning and sensitivity of the human vestibular system to low-frequency vibration. Neuroscience Letters 444: 36-41.
[69] Todd et al. 2008, p. 41.
[70] Jacob RG, Redfern MS, Furman JM. 2008. Space and motion discomfort (SMD) and abnormal balance control in patients with anxiety disorders. J Neurol Neurosurg Psychiatry, E-pub ahead of print, July 24, 2008, pp. 1-20.

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inverse activation and deactivation, such that vestibular activation deactivates visual cortex and vice versa.[71,72] In people with vestibular organ damage, long-term compensation promotes reliance on vision ("visual dependence") or on somatosensory input from muscles, tendons, joints, and skin ("surface dependence"). A visually dependent vestibular patient cannot adequately suppress visual input and up-weight vestibular signals because of pre-existing problems with the vestibular channel,[73] leaving the person dependent on visual perception of motion and position even in environments where the visual information is ambiguous. This can create fear of heights. It can also cause Space and Motion Discomfort,[74] a condition of discomfort in situations challenging to motion and position sense such as looking up at tall buildings, scanning shelves in a supermarket, closing eyes in the shower, leaning far back in a chair, driving through tunnels, riding in an elevator, riding in the back seat of a car, or reading in the car.[75]

Even without vestibular organ disease, some people have Space and Motion Discomfort due to central (brain) difficulty integrating balance signals into a coherent, moment-to-moment internal representation of position and motion. Balance testing with posturography shows that such people have difficulty down-weighting anomalous information from either the visual or somatosensory channel, or have a mild, central disorder of balance control with increased postural sway even under non-challenging conditions.[76,77,78]

Space and Motion Discomfort is common in patients with anxiety disorders,[79,80] migrainous vertigo,[81] and migraine-anxiety related dizziness.[82] Vertigo is especially characteristic of migraine and may at times occur as a migraine aura with or without headache.[83] In one study, dizziness or vertigo was found in 54%

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[71] Brandt T, Bartenstein P, Janek A, Dieterich M. 1998. Reciprocal inhibitory visual-vestibular interaction. Visual motion stimulation deactivates the parieto-insular vestibular cortex. Brain 121(Pt. 9): 1749-58.
[72] Brandt T, Dieterich M. 1999. The vestibular cortex: its locations, functions, and disorders. Ann NY Acad Sci 871: 293-312.
[73] Redfern MS, Yardley L, Bronstein AM. 2001. Visual influences on balance. J Anxiety Disord 15(1-2): 81-94.
[74] Jacob RG, Woody SR, Clark DB, Lilienfeld SO, Hirsch BE, Kucera GD, Furman JM, Durrant JD. 1993. Discomfort with space and motion: a possible marker of vestibular dysfunction assessed by the Situational Characteristics Questionnaire. J Psychopathol Behav Assess 15(4): 299-324.
[75] Jacob RG, Redfern MS, Furman JM. 2008. Space and motion discomfort (SMD) and abnormal balance control in patients with anxiety disorders. J Neurol Neurosurg Psychiatry, E-pub ahead of print, July 24, 2008, pp. 1-20. As a rural physician, I might also ask about driving past rows of parallel trees, especially with the low winter sun flashing between the trunks, as the rural equivalent of looking at lights on the wall of a tunnel.
[76] Redfern MS, Furman JM, Jacob RG. 2007. Visually induced postural sway in anxiety disorders. J Anxiety Disord 21(5): 704–16. NIH Public Access Author Manuscript, pp. 1-14.
[77] Jacob et al. 2008
[78] Furman JM, Balaban CD, Jacob RG, Marcus DA. 2005. Migraine-anxiety related dizziness (MARD): a new disorder? J Neurol Neurosurg Psychiatry 76: 1-8.
[79] Jacob et al. 2008
[80] Redfern et al. 2007
[81] Neuhauser H, Leopold M, von Brevern M, Arnold G, Lempert T. 2001. The interactions of migraine, vertigo, and migrainous vertigo. Neurology 56: 436-41.
[82] Furman et al. 2005
[83] Furman et al. 2005

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of 200 migraine patients, half of whom also had a history of motion sickness, compared with 30% of those with tension-type headaches.[84] In a study of 72 patients with isolated recurrent vertigo, 61% were found to have migraine, compared to 10% in a control group of orthopedic patients.[85] Abnormal balance testing is seen in patients with migraine but not in those with tension-type headaches.[86] Balance testing shows that both peripheral and central balance abnormalities exist in migraine patients, both more prominent if dizziness or vertigo is an associated symptom.[87]

The dizziness associated with anxiety is not necessarily created by the anxiety, as is often assumed, but may have a component of disturbed balance control.[88,89] For example, the presence of panic or fear of heights is significantly associated with abnormalities on vestibular caloric testing.[90] A positive questionnaire for Space and Motion Discomfort is significantly associated with abnormalities on posturography showing either surface[91] or visual[92] dependence. In other types of balance testing, anxiety patients have been found to have greater vestibular sensitivity than normal controls.[93] Balance assessments of patients diagnosed with panic attacks or agoraphobia (fear of leaving the house) show a high proportion with vestibular abnormalities, in some studies greater than 80%, especially if the patients have episodes of dizziness between panic attacks.[94,95,96,97]

If a person is already in a state of adaptation to ongoing vestibular or central balance deficits – even mild, fully compensated deficits – he or she is at particular risk for decompensation with exposure to new balance challenges. Many of the affected people in the present study, I suspect, were in this condition, because their medical histories reveal a variety of risks for mild baseline balance dysfunction. These risks include motion sensitivity, migraine disorder, prior damage to inner ear organs from industrial noise

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[84] Kayan A, Hood JD. 1984. Neuro-otological manifestations of migraine. Brain 107: 1123-42.
[85] Lee H, Sohn SI, Jung DK, Cho YW, Lim JG, Yi SD, Yi HA. 2002. Migraine and isolated recurrent vertigo of unknown cause. Neurol Res 24(7): 663-5.
[86] Ishizaki K, Mori N, Takeshima T,Fukuhara Y, Ijiri T, Kusumi M, Yasui K, Kowa H, Nakashima K.. 2002. Static stabilometry in patients with migraine and tension-type headache during a headache-free period. Psychiatry Clin Neurosci 56(1): 85-90.
[87] Furman et al. 2005
[88] Furman et al. 2005
[89] Eckhardt-Henn A, Breuer P, Thomalske C, Hoffmann SO, Hopf HC. 2003. Anxiety disorders and other psychiatric subgroups in patients complaining of dizziness. J Anxiety Disord 17(4): 369-88.
[90] Jacob et al. 2008
[91] Jacob et al. 2008
[92] Redfern et al. 2007
[93] Furman JM, Redfern MS, Jacob RG. 2006. Vestibulo-ocular function in anxiety disorders. J Vestib Res 16: 209-15.
[94] Perna G, Dario A, Caldirola D, Stefania B, Cesarani A, Bellodi L. 2001. Panic disorder: the role of the balance system. J Psychiatr Res 35(5): 279-86.
[95] Jacob RG, Furman JM, Durrant JD, Turner SM. 1996. Panic, agoraphobia, and vestibular dysfunction. Am J Psychiatry 153(4): 503-12.
[96] Yardley L, Britton J, Lear S, Bird J, Luxon LM. 1995. Relationship between balance system function and agoraphobic avoidance. Behav Res Ther 33(4): 435-9.
[97] Yardley L, Luxon LM, Lear S, Britton J, Bird J. 1994. Vestibular and posturographic test results in people with symptoms of panic and agoraphobia. J Audiol Med 3: 58-65.

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exposure or chemotherapy, autoimmune disease,[98] fibromyalgia,[99] normal aging (over 50), and normal early childhood.[100,101] Other potential risks for chronic vestibular dysfunction are whiplash injury and head injury, including concussions and milder head impacts without loss of consciousness.[102,103,104]

Cognition and vestibular function

It is now becoming apparent that a variety of cognitive functions depend on coherent vestibular signaling. Clinicians who work with balance-disordered patients are familiar with their struggles with short-term memory, concentration, multitasking, arithmetic, and reading.[105,106] In the perilymphatic fistula syndrome, for example (a form of inner ear pathology that can follow whiplash, minor head injuries, or pressure trauma to the ear), symptoms of dizziness, headache, stiff neck, and disturbed sleep are accompanied by marked mental performance deficiencies compared to the patient's baseline.[107] Such cognitive symptoms are difficult to evaluate clinically and are often dismissed as psychological in origin.[108] However, recent research using imaging and other modalities shows that vestibular function exerts a powerful influence over human thinking and memory.

The vestibular system is ancient in the vertebrate lineage (as reviewed above). Its neural connections ramify widely in both older and more recently evolved parts of the brain, including the brainstem, midbrain, cerebellum, and occipital, parietal, and frontal cortex.[109] Vestibular injury causes specific deficits, but not general cognitive impairment.[110] Vestibular effects on cognition are often attributed to competing stimuli (meaning challenges to movement and position sense draw attention away from

[98] Rinne T, Bronstein AM, Rudge P, Gresty MA, Luxon LM. 1998. Bilateral loss of vestibular function: clinical findings in 53 patients. J Neurol 245(6-7): 314-21.
[99] Rosenhall U, Johansson G, Orndahl G. 1996. Otoneurologic and audiologic findings in fibromyalgia. Scand J Rehabil Med 28(4): 225-32.
[100] Foudriat BA, Di Fabio RP, Anderson JH. 1993. Sensory organization of balance responses in children 3-6 years of age: a normative study with diagnostic implications. Int J Pediatr Otorhinolaryngol 27(3): 255-71.
[101] Steindl R, Kunz K, Schrott-Fischer A, Scholtz AW. 2006. Effect of age and sex on maturation of sensory systems and balance control. Dev Med Child Neurol 48(6): 477-82.
[102] Grimm RJ, Hemenway WG, Lebray PR, Black FO. 1989. The perilymph fistula syndrome defined in mild head trauma. Acta Otolaryngol Suppl 464: 1-40.
[103] Ernst A, Basta D, Seidl RO, Todt I, Scherer H, Clarke A. 2005. Management of posttraumatic vertigo. Otolaryngol Head Neck Surg 132(4): 554-8.
[104] Claussen CF, Claussen E. 1995. Neurootological contributions to the diagnostic follow-up after whiplash injuries. Acta Otolaryngol Suppl 520, Pt. 1: 53-6.
[105] Hanes and McCollum 2006
[106] Grimm et al. 1989
[107] Grimm et al. 1989
[108] Hanes and McCollum 2006
[109] Dieterich and Brandt 2008
[110] Hanes and McCollum 2006

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cognitive tasks) but may actually reflect, instead, the direct dependence of certain cognitive operations on the vestibular system.[111]

Vestibular input is critical for spatial thinking, body and spatial awareness, spatial memory, and complex spatial or map calculations.[112] Dynamic, active vestibular signaling is needed for the acquisition, storage, and use of information with spatial components, such as building mental maps or deducing a novel path between two points.[113] Patients with 5-10 year histories of bilateral vestibular loss showed marked deficits in a classic experimental task of spatial memory and navigation, accompanied, on average, by a 16.9% volume loss in the hippocampus (a temporal lobe structure essential for learning and memory).[114] In a test of general memory, however, these patients were no different from controls.[115] Vestibular signaling to the hippocampus is known to occur in both humans and other primates via a direct, two-neuron linkage through the posterior thalamus; there are also other proposed neural pathways.[116]

Disordered vestibular input increases error rates in purely mental tasks based on visualization of remembered objects, showing that coherent vestibular input is critical for thinking successfully and efficiently in spatial terms.[117] This is true even without using sight and beyond the period of memory storage. The tasks included detailed visualization, considered an occipital (visual) cortical task, and mental rotation, a parietal cortical task.[118] Vestibular stimulation in both humans and other primates activates a variety of areas in the parietal cortex, including 1) a core vestibular processing area (posterior insula), 2) the somatosensory strip, 3) areas involved in hemineglect in stroke patients (ventral parietal), 4) and a region "known to be involved in multimodal coordinate transformations and representation of space" (intraparietal sulcus), which is a principal site for arithmetic and counting tasks.[119] Hemineglect is a condition after right-sided parietal stroke in which a patient can have so much unawareness of the left side of space that he is oblivious to his own left-sided body parts being paralyzed, for example, or undressed. Vestibular stimulation temporarily corrects or improves this unawareness, in ways that suggest stimulation not only to general attention, but also to cerebral structures involved in the mental

[111] Hanes and McCollum 2006
[112] Hanes and McCollum 2006
[113] Brandt T, Schautzer F, Hamilton DA, Bruning R, Markowitsch HJ, Kalla R, Darlington C, Smith P, Strupp M. 2005. Vestibular loss causes hippocampal atrophy and impaired spatial memory in humans. Brain 128: 2732-41.
[114] Brandt et al. 2005
[115] Brandt et al. 2005
[116] Brandt et al. 2005
[117] Mast FW, Merfeld DM, Kosslyn SM. 2006. Visual mental imagery during caloric vestibular stimulation. Neuropsychologia 44(1): 101–9. I wonder whether the detailed visualization task also included a parietal component, given the quantitative comparison the subjects had to make with the remembered image.
[118] Mast et al. 2006. I wonder whether the detailed visualization task also included a parietal component, given the quantitative comparison the subjects had to make with the remembered image.
[119] Hanes and McCollum 2006, p. 82.

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representation of space.[120,121] Vestibular stimulation also improves hemineglect patients' performance on tasks of visual localization and visual-spatial memory retrieval. At baseline, and again 24 hrs after the experiement, their responses were biased away from the left side, but this bias was corrected or improved immediately after left vestibular stimulation.[122]

Studies of hemineglect patients have further shown that many mental operations are "spatialized" and dependent on parietal brain areas that have been lost, including mathematical operations involving a "mental number line" with lower numbers on the left,[123,124] clock representations of time,[125] and spelling at the beginnings (left) or ends (right) of words (errors occur opposite to the side of the parietal lesion).[126] In right-handed patients with right parietal strokes, there is no impairment to simple numeric calculation (a left-sided parietal function), but there is impairment to spatialized mathematical thinking, such as finding the midpoint between two numbers.[127] At the other extreme of mental functioning, it has been found that great mathematicians think of numbers in spatial terms,[128] which "may be more efficient because it is grounded in the actual neural representation of numbers."[129] A recent study of outstanding human memorizers shows that spatially oriented strategies are also critical to good memory, by providing an efficient framework for memory organization and retrieval.[130]

In summary, coherent vestibular neural input is critical for spatialized forms of thinking and memory. Spatialized thinking and memory is intrinsic to many of the things we do with our minds, including mathematical thinking and memory organization (as discussed above) and many forms of map-based or visually based problem-solving or short-term memory we do in everyday life. Spatial thinking is used, for example, to figure out the most efficient path for a set of errands, remember the path and images of the items to be obtained, search for the items on the shelf, and judge if one was given the correct change. It is used for mental "maps" or calendars of one's day, week, or month and its appointments, to picture in three dimensions how to put something together, or imagine what has gone wrong inside a device and initiate a

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[120] Geminiani G, Bottini G. 1992. Mental representation and temporary recovery from unilateral neglect after vestibular stimulation. J Neurol Neurosurg Psychiatry 55(4): 332-3.
[121] Cappa S, Sterzi R, Vallar G, Bisiach E. 1987. Remission of hemineglect and anosognosia during vestibular stimulation. Neuropsychologia 25: 775-82.
[122] Geminiani and Bottini 1992
[123] Zorzi M, Priftis K, Umilta C. 2002. Brain damage: Neglect disrupts the mental number line. Nature 417: 138-9.
[124] Vuilleumier P, Ortigue S, Brugger P. 2004. The number space and neglect. Cortex 40(2): 399-410.
[125] Vuilleumier et al. 2004
[126] Hillis HE, Caramazza A. 1995. Spatially specific deficits in processing graphemic representations in reading and writing. Brain Lang 48 (3): 263-308.
[127] Zorzi et al. 2002
[128] Hadamard J. 1996. *The Mathematician's Mind: The Psychology of Invention in the Mathematical Field.* Princeton University Press, NJ. *In* Zorzi et al. 2002.
[129] Zorzi et al. 2002
[130] Maguire EA, Valentine ER, Wilding JM, Kapur N. 2003. Routes to remembering: the brains behind superior memory. Nat Neurosci 6(1): 90-5.

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repair. It is used for understanding the images in a movie or TV show with the sensitivity not to miss subtle visual clues. In this context, it is easy to see how vestibular disturbance could impact concentration (which means the ability to perform thinking tasks successfully and efficiently) and memory. Vestibular disturbance also has the potential to affect reading directly via the reflex control exerted by semicircular canal and otolith organs over eye movements (vestibulo-ocular reflex).

Effects on concentration and memory were nearly ubiquitous in the present study, if one includes all subjects that told me about any problem in this area. For some subjects the deficits were dramatic compared to baseline (before exposure), including the 7 out of 10 school-age children and teens who showed a decline in their academic performance. Detrimental effects on concentration and memory were significantly associated with normal memory at baseline ($p = 0.027$) and with fatigue and loss of energy and motivation during exposure ($p = 0.018$). Though sleep deprivation and disturbance undoubtedly play a role, qualitative aspects of the mental performance deficiencies suggests a mechanism other than sleep disturbance alone. I propose that this mechanism is the effect of vestibular disturbance on cognition.

It is interesting here to examine a possible role of vestibular disturbance in the learning of very young children, in the toddler and preschool years. Mrs. G. (G2) volunteered that her 2½-year-old's (G5) irritability during turbine exposure was especially triggered by her older siblings' "unsteadying her" or coming so close that she thought she might be unsteadied. Children at this age are learning to keep their balance through a variety of different kinds of activities and postures. They are both fascinated and relaxed by vestibular stimulation (swinging, spinning, rolling, somersaults, etc.) and they actively explore the physical world through this play. The behavior of objects in gravity is another source of fascination, starting with babies' casting behavior and moving on to pouring water, sliding down slides, rolling things down inclines, building dams, floating toy boats, blowing bubbles, releasing helium balloons, etc. Vestibular input and processing play a critical role in balance during movement; in the generation, storage, and use of internal maps; and in recognition of the behavior of objects under the influence of gravity. Indovina et al. measured brain activity by functional MRI while adult subjects watched the movement of simulated objects, finding that the vestibular network was selectively engaged when the acceleration of the object was consistent with natural gravity, even though the stimulus was only visual.[131] The authors use this as evidence that "predictive mechanisms of physical laws of motion are represented in the human brain"[132] under the influence of vestibular signaling of the vector of gravity. I suggest that these representations of the physical laws of motion are embedded in the human brain during early

[131] Indovina I, Maffei V, Bosco G, Zago M, Macaluso E, Lacquaniti F. 2005. Representation of visual gravitational motion in the human vestibular cortex. Science 308: 416-9.
[132] Indovina et al. 2005

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childhood as toddlers and children learn through experimentation (play) about the behavior of their bodies and other objects in gravity, and that coherent vestibular signaling is critical to this learning.

Environmental noise, learning, sleep, and health effects

Many studies have quantified the effects of environmental noise on children's learning. Reading acquisition – a language-intensive process – is especially sensitive to the effects of noise in school and at home. The effect is distinct from the effects of noise on attention or working memory,[133] and is correlated with measures of language processing such as speech recognition.[134] Airplane noise, which has a large low frequency component, has a stronger effect than traffic noise in some studies,[135] but traffic noise is also shown to have modest effects on memory in quieter communities.[136] Most studies are cross-sectional, but a longitudinal or cohort study, done when an airport was closed in one location and opened in another, showed similar effects on reading acquisition.[137] One study showed effects of noise on reading and auditory processing in children who lived in an apartment building next to a busy highway. The higher they lived in the building, the quieter were their apartments and the better their reading and auditory discrimination scores (e.g., distinguishing *goat* from *boat*). After controlling for parental education and income, the auditory discrimination scores largely explained the noise-reading linkage.[138] These effects on reading occur at sound levels far less than those needed to produce hearing damage.[139] Children with pre-existing reading deficiencies and children at higher grade levels are more affected, and longer exposure produces larger deficits.[140]

Effects suggestive of wind turbine noise impact on auditory discrimination or central auditory processing were found in the current study. During the period immediately after moving away from turbines and the cessation of her tinnitus, Mrs. A (A2, age 33) found she had a new difficulty understanding conversation

[133] Haines MM, Stansfeld SA, Job RFS, Berglund B, Head J. 2001. A follow-up study of effects of chronic aircraft noise exposure on child stress responses and cognition. International Journal of Epidemiology 30: 839-45.
[134] Evans GW, Maxwell L. 1997. Chronic noise exposure and reading deficits: the mediating effects of language acquisition. Environment and Behavior 29(5): 638-56.
[135] Clark C, Martin R, van Kempen E, Alfred T, Head J, Davies HW, Haines MM, Barrio IL, Matheson M, Stansfeld SA. 2005. Exposure-effect relations between aircraft and road traffic noise exposure at school and reading comprehension: the RANCH project. Am J Epidemiol 163: 27-37.
[136] Lercher P, Evans GW, Meis M. Ambient noise and cognitive processes among primary schoolchildren. Environment and Behavior 35(6): 725-35.
[137] Hygge S, Evans GW, Bullinger M. 2002. A prospective study of some effects of aircraft noise on cognitive performance in schoolchildren. Psychol Sci 13: 469-74.
[138] Cohen S, Glass DC, Singer JE. 1973. Apartment noise, auditory discrimination, and reading ability in children. Journal of Experimental Social Psychology 9: 407-22.
[139] Evans GW. 2006. Child development and the physical environment. Annu Rev Psychol 57: 423-51.
[140] Evans 2006, p. 426.

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superficial. The results also suggest that the health status sampling was inadequate. For example, the study found that only 2% of respondents indicated they had chronic migraine disorder,[189] whereas the population prevalence is probably 12-13%. Likewise, tinnitus prevalence in this study was 2%, whereas 4% is a more likely population figure for the average age of 54.[190] Tinnitus prevalence was not correlated with age in this sample,[191] while in reality tinnitus has a well-documented pattern of increasing prevalence with advancing age.[192] In sum, this survey did not adequately sample health status, and was not designed with comparison groups so as to detect differences.

Recommendations

For physicians practicing near wind turbine installations, I suggest incorporating proximity to turbines into the personal and social history in a neutral and non-suggestive way, especially for the types of symptoms described in this report.

With regard to turbine setback from dwellings: In Table 1B we see that the subjects in the current study lived between 305 m (1000 ft) and 1.5 km (4900 ft or 0.93 mi) from the closest turbine. There were three severely affected families at 930-1000 m (3000-3300 ft) from turbines. This study suggests that communities that allow 1000-1500 ft (305-457 m) setbacks from houses may have families who need to move after turbines go into operation. All turbine ordinances, I believe, should establish mechanisms to ensure that turbine developers will buy out any affected family at the full pre-turbine value of their home, so that people are not trapped between unlivable lives and destitution through home abandonment. By shifting the burden of this expense to turbine developers, I would hope that developers might have a stronger incentive to improve their techniques for noise prediction and to accept noise level criteria recommended by such agencies as the World Health Organization and the International Standards Organization,[193] and fortified by the findings of Pedersen and van den Berg (above). With regard to families already affected, developer and permitting agencies share the responsibility for turbines built too close to homes, and together need to provide the financial means for these families to reestablish their lives at their previous level of health, comfort, and prosperity.

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[189] van den Berg et al. 2008b, p. 48.

[190] National Institute on Deafness and Other Communication Disorders, USA, website, "Prevalence of Chronic Tinnitus." 2009. http://www.nidcd.nih.gov/health/statistics/prevalence.htm

[191] van den Berg et al. 2008b, p. 47.

[192] National Institute on Deafness and Other Communication Disorders, USA, website, "Prevalence of Chronic Tinnitus." 2009. http://www.nidcd.nih.gov/health/statistics/prevalence.htm

[193] See George Kamperman and Richard James, *The "How To" guide to siting wind turbines to prevent health risks from sound,* 47 pp., at http://www.windturbinesyndrome.com/?p=925 (2008).

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I support the recommendations for noise level criteria and procedures for noise monitoring by George Kamperman and Richard James.[194] A single setback distance may not be both protective and fair in all environments with all types of turbines, but it is clear, from the current study and others, that minimum protective distances need to be more than the 1-1.5 km (3280-4900 ft or 0.62-0.93 mi) at which there were severely affected subjects in this study, more than the 1.6 km (5250 ft or 1 mi) at which there were affected subjects in Dr. Harry's UK study,[195] and, in mountainous terrain, more than the 2-3.5 km (1.24-2.2 mi) at which there were symptomatic subjects in Professor Robyn Phipps's New Zealand study.[196] Two kilometers, or 1.24 miles, remains the baseline shortest setback from residences (and hospitals, school, nursing homes, etc.) that communities should consider. In mountainous terrain, 2 miles (3.2 km) is probably a better guideline. Setbacks may well need to be longer than these minima, as guided by the noise criteria developed by Kamperman and James.

Suggestions for further research

- Epidemiologic studies comparing populations exposed and not exposed to wind turbines with regard to the prevalence of specific symptoms, such as tinnitus and balance disorders. Such studies might be best conducted in European countries that have both national health data systems and significant numbers of wind turbines.

- Case series by neurotologists internationally, who are able to do appropriate objective examination and testing in addition to clinical history.

- Collaboration between physicians and independent noise engineers to correlate specific frequencies and intensities of sound and vibration with subjects' symptoms, and to establish a standard protocol for noise sampling that captures the elements relevant to health effects.

- Further clinical/laboratory research on the effects of low frequency noise and vibration on the human vestibular system.

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[194] Kamperman GW, James RR, *The "How To" guide to siting wind turbines to prevent health risks from sound*, 47 pp., at http://www.windturbinesyndrome.com/?p=925 (2008). Presented in shorter form, Kamperman GW, James RR, "Simple guidelines for siting wind turbines to prevent health risks," at the annual conference of the Institute of Noise Control Engineering/USA, Noise-Con, July 28-31, 2008.

[195] Harry, Amanda, 2007.

[196] Phipps, Robyn, 2007.

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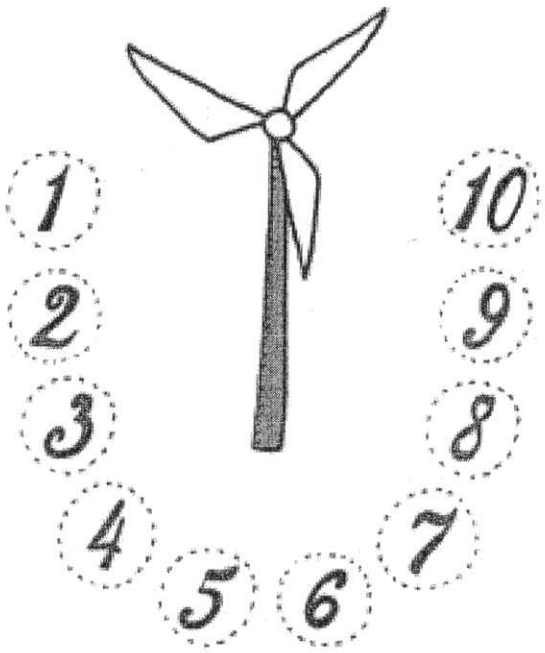
Wind Turbine Syndrome for Non-Clinicians

Abstract

I interviewed 10 families living near large (1.5 to 3 MW) wind turbines, all of which were built since 2004. This gave me 38 people, from infants to age 75. Their symptoms formed a cluster (see Glossary for clinical terms):



1) sleep disturbance
2) headache
3) tinnitus (pronounced "tin´-ah-tus": ringing or buzzing in the ears)
4) ear pressure
5) dizziness (a general term that includes vertigo, lightheadedness, sensation of almost fainting, etc.)
6) vertigo (clinically, vertigo refers to the sensation of spinning, or the room moving)
7) nausea
8) visual blurring
9) tachycardia (rapid heart rate)
10) irritability
11) problems with concentration and memory
12) panic episodes associated with sensations of internal pulsation or quivering, which arise while awake or asleep

The families not only lived near turbines and developed new symptoms; they moved away from the turbines (because they were so troubled, often abandoning their homes) and the symptoms, significantly, went away.

Hence, the definitive result of my report is that wind turbines cause the symptoms of Wind Turbine Syndrome (WTS).

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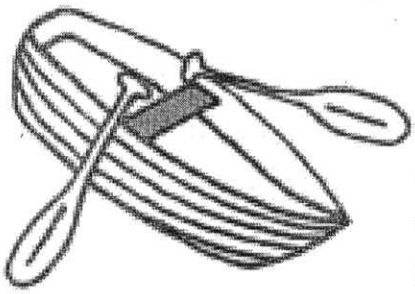
Let's clarify something immediately. Not everyone living near turbines gets these symptoms. As a solo, unfunded researcher I could not get the samples needed to figure out what percentages of people at what distances get the symptoms. This needs to be done next. But I could (and did) look at the question of why some people are susceptible and others not, plus who is susceptible, and I used these patterns to explore the pathophysiology of Wind Turbine Syndrome: what's going on inside people to cause these specific symptoms.

I would like readers to be able to look at this study—including the detailed accounts I provide of people's experiences around turbines and their health backgrounds—and be able to make their own decisions about whether they should be exposed to these machines.

That said, I was able to prove mathematically that people with pre-existing migraines, motion sensitivity (such as car-sickness or seasickness), or inner ear damage are especially vulnerable to these symptoms. Equally as interesting, I was able to demonstrate that people with anxiety or other preexisting mental health problems are not especially susceptible to these symptoms.

This contradicts wind industry literature, which argues that people who worry about or otherwise dislike turbines ringing their homes are the ones getting ill. I show this to be complete nonsense.



Here is what's going on, as I put together the evidence. *Low frequency noise tricks the body's balance system into thinking it's moving.* Like seasickness. (It's vital to understand that the human balance system is a complex brain system receiving nerve signals from the inner ear, eyes, muscles and joints, and inside the chest and abdomen. Because the eyes are involved, visual disturbance from the blades' shadow flicker adds to the balance disturbance.)

Let me repeat this, because its significance is huge. *Low frequency noise from turbines appears to deceive the body into thinking it's moving.* So what, you say? Not so fast! Research within the last 10 years has demonstrated conclusively that *the way our bodies register balance and motion directly affects an astonishing array of brain functions.*

How? By direct neurologic linkages connecting the organs of balance to various, seemingly unrelated brain functions.

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